a normal fetal development. A healthy male baby weighing 2,770 g was delivered vaginally at term without obstetric complications. The child has continued a normal development.

IFN-α has been used at diverse range of dosage for disease control during pregnancy in women with several myeloproliferative disorders and hairy cell leukemia without teratogenic or abortive effects reported [3,5].

Our experience suggests that young women of childbearing age with ET who are willing to become pregnant might undergo an IFN-α trial ahead of time to confirm their response to the drug and to find a temptative dose necessary to initially control the disease. IFN-α can then be administered during a subsequent pregnancy, adjusting the dosage to maintain an appropriate platelet count until delivery.

We did not observe any harmful IFN-α effect in the fetus, but the indications and safe use of IFN in the management of pregnant ET patients await further cumulative experience.

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Acquired Factor V Inhibitor in a Patient With Acquired Human Immunodeficiency Syndrome

To the Editor: Autoimmune syndromes have been described in patients with human immunodeficiency virus (HIV) infection. Examples include autoimmune thrombocytopenic purpura (AIT) and antineuronal antibody-mediated neuropathies [1]. Acquired inhibitors have been reported with

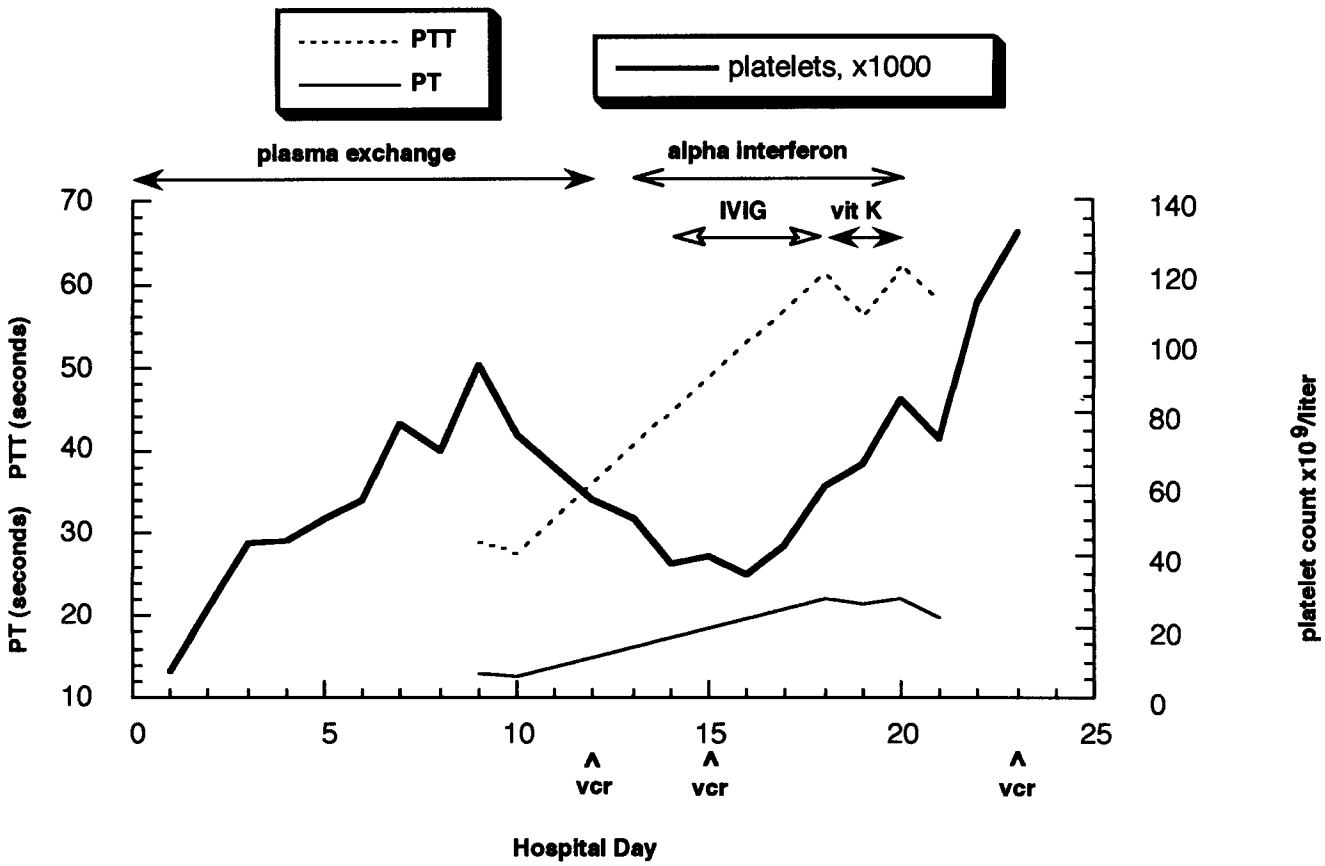

Fig. 1. Coagulation indices and treatment versus hospital day. Prothrombin time (PT), partial thromboplastin time (PTT), and platelets as a function of hospital stay. Plasma exchange was 14 units of FFP daily for days 1–6 and 12 units daily for days 7–12. IVIG dose was 24,800 mg every 24 hours days 14–18. Interferon-α dose was 3 million units sc day 13, 6 million units sc day 14, and 9 million units sc days 15–20. Vincristine (vcr) dose was 1 mg IV on days 12, 15, and 23. Vitamin K (vit K) dose was 10 mg IM days 18–20.

plasma and blood transfusions, infections, autoimmune disorders, pregnancies, malignancies, and antibiotics [2–4]. We report the first case of an acquired factor V inhibitor in the setting of HIV infection.

A 32-year-old man with HIV infection presented 10 days prior to admission with fever and weight loss for 2 months and diffuse lymphadenopathy. His hemoglobin (Hb) and platelet count were normal with a CD4 count of 220/μl (nl 410–1,840) and a CD8 count of 1730/μl (nl 270–870). On admission, he had a temperature of 100.5°F, and a Hb of 82 g/L (nl 120–150), platelet count of 7×10^9/L (nl 150–450), reticulocyte % of 3.9 (nl 0.5–1.5), and LDH of 1,725 U/L (nl 297–611). Prothrombin time (PT), international normalized ratio (INR), and partial thromboplastin time (PTT) were normal. A direct Coomb's antibody test was negative. The peripheral smear contained schistocytes and large platelets. His mental status, creatinine, and urinalysis were normal. A diagnosis of thrombotic thrombocytopenic purpura (TTP) was made and plasma exchange was begun. After initial improvement (Fig. 1), the platelet count decreased. A bone marrow biopsy showed plasmacytosis and increased megakaryocytes. Elevated antiplatelet antibodies [IgM 19.7% (nl 2.2–7.2) and IgG 37.4% (nl 2.1–12.1)] suggested a diagnosis of AIT. Plasma exchange was discontinued, and intravenous immunoglobulin (IVIG) was started. A cervical lymph node showed Kaposi sarcoma (KS), and an abdominal CT scan showed lymphadenopathy and splenomegaly. His thrombocytopenia was thought due to HIV-associated AIT and splenomegalic-KS, so he was treated with azidothymidine and vincristine plus interferon-α (IFN-α). A preoperative evaluation for a splenectomy revealed a PT of 21.9 sec (nl 11.1–12.9), INR of 4.3 (nl ratio 1.0), and PTT of 61.2 sec (nl 20.0–33.5). These did not correct with vitamin K or fresh frozen plasma. His measured factor II activity was 64% (nl 83–126), factor V was 12% (nl 66–152), factor VII was 59% (nl 66–156) and factor X was 75% (nl 65–142). A factor V mixing study was 9% (nl 85%) and factor V 50:50 mixed study was 28% (nl 47%), demonstrating the presence of a factor V inhibitor. Splenectomy was postponed, and on hospital day 17 his platelet count increased. He was discharged home with a platelet count of 131×10^9/L. His coagulation indices normalized 1 month later while receiving weekly vincristine.

During plasma exchange, he was exposed to 156 units of foreign factors that likely served as the antigenic stimulus to develop the inhibitor. Patients with coagulation factor inhibitors have been treated with FFP, cyclophosphamide, and prednisolone; platelets are used specifically for factor V inhibitors [5]. Specific therapy was not instituted because the patient was asymptomatic, the risk of further immunosuppression, and the possibility his inhibitor might disappear spontaneously. His asymptomatic course suggests that expectant management may be appropriate.

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Leukopenia, Thrombocytopenia, and Acute Autoimmune Hemolytic Anemia Associated With an Unusual (Type 2/4) Hodgkin's Disease: Case Report

To the Editor: The association of autoimmune hemolytic anemia (AIHA) and Hodgkin disease (HD) was first described in 1967 [1]. The reported frequency of this association ranges from 0.2% [2] to 1.7% [3]. Autoimmune neutropenia is extremely uncommon in HD [4,5], in contrast to immune thrombocytopenia (ITP) which is more frequent in HD (1% to 2%) than in the general population [2]. We describe here a new presentation of HD, associating AIHA, ITP and leucopenia.

A 24-year-old woman was admitted for asthenia, jaundice, and fever. Physical examination discovered cervical adenopathies, splenomegaly, and hepatomegaly. Computed tomography (CT) detected mediastinal, celiomesenteric and lomboaortic adenopathies. Hemoglobin level was 4 g/dl; platelets, 71,000/mm^3, and white blood cells (WBC), 1,300/mm^3, with 74% neutrophils, 14% lymphocytes, and 12% monocytes. The haptoglobin level was 0.06 g/L (normal value 0.54–1.43), total bilirubin was 65 μmo/L (normal value <20), mainly unconjugated (41 μmol/L), lactate dehydrogenase (LDH) was 411 IU/L (normal value <330). The reticulocyte count was 394,000/mm^3. The direct and indirect Coombs tests were positive for IgG, but not for complement. Fibrinogen was 3 g/dl, C-reactive protein (CRP) was 24 mg/L (normal value <6), coagulation tests were normal; and no schizocytes were observed. The human immunodeficiency virus (HIV) test was negative. Hemoglobin electrophoresis was normal. The serum electrophoresis showed polyclonal immunoglobulin increase, but without any monoclonal component on immunoelectrophoresis. The medullogram was hypercellular, showing an important erthrocytic hyperplasia (61%), a rich granulocytic lineage (28%), and many megacaryocytes. Osteomedullar biopsy revealed foci of neoplastic involvement containing few atypical large cells surrounded by fibrosis, suggesting HD lesions. A large cervical lymph node was excised and histological analysis (Fig. 1) showed replacement of the normal node architecture by widespread fibrosis containing a few nodular nests of small lymphocytes and scattered Reed-Sternberg cells (R-SC) of classic immunophenotype (CD15+ and CD30+, CD3−). The lymphocytic cellular component exhibited marked plasmocytoïd features (Fig. 1C) with a polyclonal pattern of intracytoplasmic Ig light chain immunodetection. The diagnosis of HD was done, as an intermediate form between type 2 (nodular sclerosis) and type 4 (lymphocyte depleted, fibrotic). Acute intravascular AIHA lead to corticosteroid treatment (2 mg/kg/day) that had no results after 10 days. The severity of anemia prompted plasmapheresis, which was partly successful; the platelets increased to 152,000/mm^3, WBC to 6,100/mm^3, and hemoglobin to 6.5 g/dl. Unfortunately, this response was only transient, and finally, splenectomy was performed, that obtained only transient cessation of hemolysis, with little effect on platelet and leukocyte numeration. Histological examination of the spleen found HD lesions, in association with myeloid metaplasia. A chemotherapy (MOPP regimen) was initiated, but the patient died on day 10 of sepsis and disease progression.

Severe pancytopenia at diagnosis rised the problem of chemotherapy beginning. The attempts to resolve the autoimmune process (corticosteroids, plasmapheresis, splenectomy) gave only mild and transient results, leading to chemotherapy despite the hematological conditions. Although the issue of this patient was unfavourable, salvage chemotherapy was the only solution since the prognosis of cytopenias seems to be related only to the status of the underlying HD [2].

The autoimmune origin of the hemolytic anemia is proven by the positive Coombs test. The peripheral origin of thrombocytopenia is supported by the presence of numerous megacaryocytes in the marrow, and its immunological mechanism was suggested in the absence of other causes of platelet destruction (e.g., viral infection, disseminated intravascular coagulation). Although hypersplenism could have participated, splenectomy did not improve the thrombocytopenia. The peripheral origin of neutropenia was also most